

Malnutrition-induced cataracts in an orphaned kitten

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A 0.75-kg, approximately 9-week-old, male grey tabby was presented for general malaise. The owner reported that the kitten was notably less active than it had been 1 wk prior when she had adopted it from a local humane society. According to the owner, it had been orphaned and raised on a commercial kitten milk replacer. At the time of presentation, it had been weaned for approximately 2 wk and was eating feline growth diet. At the time of adoption, the kitten's stools had been loose but had recently normalized. The owner had not observed any vomiting. The kitten had been treated with pyrantel pamoate (10 mg/kg bodyweight (BW), PO) 2 and 3 wk prior to presentation; it also received its first multivalent vaccination for feline viral rhinotracheitis, calicivirus, and parvovirus at the same time as the first dose of anthelmintic.

At presentation, the kitten appeared thin and had a temperature of 39.7°C. A cursory ophthalmologic examination revealed bilateral lens opacification, the extent of which could not be fully appreciated without chemical dilation of the pupils. There were no other abnormal findings on physical examination.

The pupillary light and menace responses were normal bilaterally. The kitten was able to negotiate obstacles and visually follow moving objects. The pupils dilated normally following the administration of 1 drop of tropicamide 1% ophthalmic solution to each eye. Ophthalmologic examination revealed extensive, bilaterally symmetrical, dense lens opacification of the posterior cortex. Symmetrical, narrow, 3-pronged, linear areas representing the suture lines of the posterior cortices appeared unaffected. The remainder of the ophthalmologic examination, including intraocular pressures and the appearance of the adnexa, cornea, anterior chamber, and the portions of the retina that could be seen through the transparent areas of lens, were within normal limits.

The owner was instructed to move the kitten's pen out of the sun to a cooler location; to monitor his temperature, appetite, and attitude; and to closely observe him for signs of blindness. A phone conversation 2 d following the presentation elicited that the kitten seemed completely normal.

Two weeks later, when the kitten presented to the clinic for its booster vaccinations, ophthalmological examination revealed that the lens opacities appeared

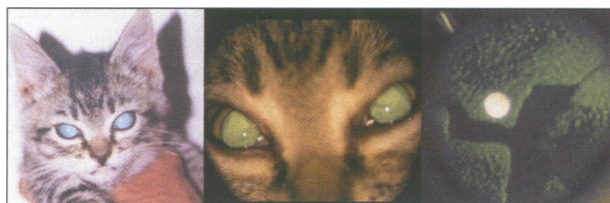


Figure 1. Bilateral cataracts in an 11-week old kitten. Pupils are chemically dilated with tropicamide 1% ophthalmic solution. Lesions are characterized by extensive, bilaterally symmetrical, dense lens opacification of the posterior cortex. Symmetrical, narrow, 3-pronged, linear areas representing the suture lines of the posterior cortices appeared unaffected.

less dense and had receded away from the suture line (Figure 1). Five weeks following presentation, only a 3-mm white opacity was present on the ventronasal equator of the left lens, and by 12 wk following presentation, the cataracts had completely resolved.

The posterior cortical opacities were most likely a result of intercellular fluid accumulation between the posterior cortical lens fibers. Such "water clefts" or vacuoles are often due to a disruption of lens metabolism caused by an alteration in the biochemical composition of the aqueous humor. A common example of this is the early diabetic cataract. In this case, resolution of the lesions following presentation indicates that whatever caused the alteration of the lens metabolism had corrected itself before precipitation of lens proteins caused permanent opacification.

Primary and congenital cataracts are rare in the cat. Most are secondary, associated with trauma, anterior uveitis, and lens luxations. There have been reports, however, of metabolic cataracts in kittens associated with nutritional secondary hyperparathyroidism and hypocalcemia, feline diabetes (1), and kittens fed commercially available kitten milk replacer (2). The latter, a study comparing kittens fed commercial milk replacer (CMR) with those fed queen's milk, documented the development of watery, yellow diarrhea within 48 h of feeding a CMR, as well as the development of lens opacification within 2 wk. The ocular lesions were characterized by increased prominence, feathering, and vacuolization of the posterior Y-suture, as well as diffuse anterior and posterior cortical opacification. By 4 wk, the ocular lesions had worsened in 4 of the 5 kittens. Within 4 wk of replacing the CMR diet with a feline growth formula, the lens opacities resolved to a residual perinuclear halo and a few incipient cortical opacities.

Amino acid-deficient diets have been associated with cataract formation in many species, including canids

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(3–5), rats (6), guinea pigs (7), swine, and salmonids (8). In the kitten study cited above (2), the serum arginine concentration in the CMR-fed kittens was one-third lower (132.2 nmol/mL) than the queen's milk-fed kittens (311.5 nmol/mL). This was most likely a result of the lower arginine content of the CMR (129.8 μ mol/g of dry matter) as compared with that of queen's milk (253.9 μ mol/g of dry matter). Based on the comparative nutritional profiles of the diets, serum amino acid concentrations, and resolution of lens opacities following weaning, the study concluded that arginine deficiency may cause cataracts in kittens.

Several commercial kitten milk replacers are currently being marketed. However, no guidelines have been set forth by the Association of American Feed Control Officials for testing milk replacers prior to marketing (2). Some companies have added L-arginine to their formulation (2). Based on the history and clinical presentation of the kitten being reported here, it may be that the cataracts were secondary to hypoargininemia caused by a milk replacer diet deficient in arginine.

Veterinarians should routinely perform ophthalmological examinations on kittens and be aware of the possibility of nutritionally induced cataract formation.

Furthermore, they should compare nutritional profiles of available kitten milk replacers and avoid those lower in arginine.

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BOOK REVIEW



COMPTE RENDU DE LIVRE

Kesel ML. *Veterinary Dentistry for the Small Animal Technician*. Iowa State University Press, Ames, Iowa, 2000, 280 pp, ISBN 0-8138-2037-5, US \$34.95

This book covers most aspects of dentistry that a veterinary technician needs to know, from understanding the basics to performing a dental prophylaxis, charting oral lesions and assisting advanced dental procedures. The technician's role is very well explained, which should help them become more efficient as dental team members. I did, however, find that the technical information is not always accurate or complete.

The content of the book is well organized. Starting with oral and dental anatomy and then moving through equipment maintenance, dental radiology, periodontal pathology, and dental prophylaxis is a logical progression for the reader. Anesthesia and analgesia are addressed in Chapter 4, which is followed by a chapter on dental charting, which includes more notions of pathology, periodontal and dental indices, etc.

About one third of the book is dedicated to advanced dental procedures. They are grouped by disciplines: exodontics, periodontics, endodontics, orthodontics, prosthodontics, and restorative dentistry. It is interesting that these procedures are discussed from the point of view of the technician assisting the veterinarian. This insight should help the technician and veterinarian work as a team and improve the quality of their work.

The last chapter is about client education and it includes a collection of handouts that inform the client about the pathology, diagnostic, treatment and aftercare. The appendices on related books, veterinary dental associations, abbreviations, position statement of the American Veterinary Dental College and a list of dental suppliers provide useful information.

The author uses more of an everyday than a purely medical language in her book. She explains medical and dental concepts by making analogies to ordinary life. Even if these analogies may sometimes lack exactitude, they have the benefit of making the concepts easier to understand for newcomers to the field.

This book contains a lot of information. The quality of the information, though, is not constant. At times, the information is inaccurate. For example, technicians are wrongly advised that a power scaler should be "cranked to the most powerful setting in order to clean off calculus faster." There are too many of these inaccuracies in this book to recommend it as the main or only source of dental information for a technician.

In my opinion, this book's value is in its description of the role of the technician and the popularization of many dental concepts.

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